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DNA METHYLATION IN PLANTS

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Common DNA methylation controls gene expression and preserves genomic integrity. Mal methylation can cause developmental abnormalities in the plants. Multiple enzymes carrying out de novo methylation, methylation maintenance, and active demethylation culminate in a particular DNA methylation state. Next-generation sequencing advances and computational methods to analyze the data. The model plant Arabidopsis thaliana was used to study DNA methylation patterns, epigenetic inheritance, and plant methylation. Plant DNA methylation research reveals methylation patterns and describing variations in plant tissues. Determining the kinetics of DNA methylation in diverse plant tissues is also a new field. However, it is vital to understand regulatory and developmental decisions and use plant model species to develop new commercial crops; that are more resistant to stress and yield more. There are several methods available for assessing DNA methylation data. The performance of several techniques is assessed in A. thaliana, which has a smaller genome than hexaploid bread wheat.
Keywords: DNA methylation, plants, process, use and benefits.

INTRODUCTION

DNA methylation is a biological process that adds methyl groups to DNA. Methylation can modify a DNA region's activity without changing the sequence, and DNA methylation at a gene promoter generally inhibits transcription. The methylation of cytosine at position 5 (also known as 5meC) is an epigenetic modification (Costello *et al.,* 2001). No matter how much and where DNA methylation occurs, all plant genomes studied so far exhibit substantial 5meC methylation (Takuno *et al.,* 2016; Bewick *et al.,* 2016). The model plant Arabidopsis thaliana has facilitated us to understand DNA methylation. Plants need methylation of repetitive sequences and transposons on DNA to block transcription and create heterochromatic areas. Mutations that reduce DNA methylation cause transposon activation and genomic breakdown in Arabidopsis thaliana after a few generations.

The biological role of DNA methylation in virus resistance and plant development was first identified in 2009 (Buchmann *et al.,* 2009; Castillo *et al.,* 2015; Feng *et al.,* 2010, Rodriguez *et al.,* 2013, Wang *et al.,* 2014; Yang *et al.,* 2011). Early generations can outcross the mutation, resulting in isogenic offspring with different DNA methylation states (Lauss *et al.,* 2018; Kooke *et al.,* 2015). Similarly, studies have demonstrated that alterations in DNA methylation can affect ecologically relevant phenotypic characteristics (Hauser *et al.,* 2011; Quadrana *et al.,* 2016). Unlike animals, plants maintain 5meC in various sequence situations (CG, CHG, and CHH, where H can be any bases A, C, or T) catalyzed by numerous methyltransferases. On the other hand, in A. thaliana, CMT3 maintains CHG methylation, whereas CMT2 maintains CHH methylation. CG methylation occurs in both euchromatin and heterochromatin (Deleris *et al.,* 2012). The redundant and cross-functional DNA methylation processes assist in identifying invading transposons and permanently stop their synthesis (Kim *et al.,* 2012). It is possible that single-repeat retroelements that may swiftly amplify via reverse transcription or DNA transposons that utilize a copy-and-paste approach to amplify during DNA replication cause variations in repeat content in plants (Wicker *et al.,* 2007; Tsukahara *et al.,* 2009). While Arabidopsis has a 20% repetition rate, cereals like barley and wheat may have up to 90% repetition. Because hexaploid

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wheat has three subgenomes, these repetitions demand tightly controlled epigenetic processes (Eichten *et al.,* 2016).

***DNA Methylation Mechanism*:** DNA methylation is an epigenetic mark that controls gene expression, genomic stability, and gene imprinting. DNA methylation is a conserved epigenetic control that includes adding a methyl group to cytosines and is involved in TGS and transposon taming. Transcriptional regulation relies on DNA methylation (Castillo *et al.,* 2013; Law *et al.,* 2010). Transposable elements and other repetitive DNA sequences in plants are heavily methylated, required for transcriptional silence (Cokus *et al.,* 2008; Lister *et al.,* 2008). In Arabidopsis thaliana, methylation and demethylation act as a disease defense. These processes are essential for disease resistance in plants. It also controls immune transcription and co-transcription.

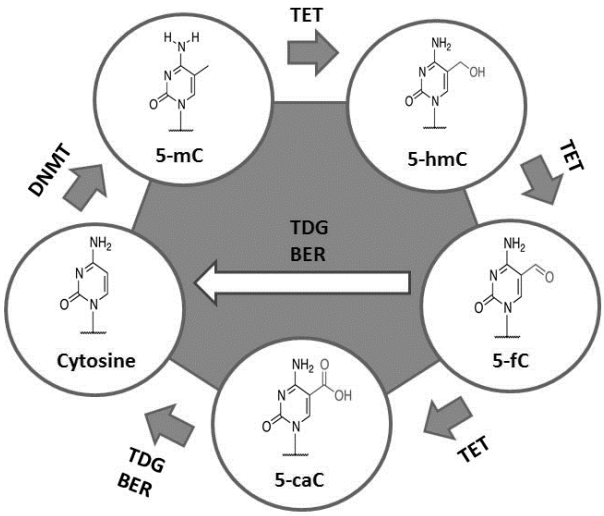

Figure 1. Depiction of cytosine's methylation and demethylation processes.

A transposon-linked defense gene is produced when the immune system's dynamic methylation changes. Plants' chemical signals are connected to their immune system. Pattern-recognition receptors identify conserved microbial fingerprints. This recognition prompts an immune response. The first plants to identify conserved microbial fingerprints were rice (XA21, 1995) (Ronald and Beutler, 2010). Plant immune receptors detect pathogen effectors. One is the NBS-LRR protein family. When a plant interacts with a pathogen, it has a bad hypersensitive reaction. Hypersensitivity causes localized cell death at the injection site (HR). It prevents the plant from being infected and spreading disease. A cysteine protease controls cell disintegration in mammalian proptosis following death (Rojo *et al.,* 2004). Plants have resistance genes expressed by resistant proteins that detect pathogens. These proteins' domains resemble toll-like and nod-like receptors in animals' immunity. Methylation is an epigenetic alteration that happens in DNA bases. It is found at the fifth carbon of the cytosine pyrimidine ring in mammals and plants. Methylation may inhibit transposons, affecting gene transcription. Stressors can alter methylation status at particular loci, although it is mainly hereditary. It is a CG scene in animals. Non-methylated CG is present in embryonic stem cells and neurons (Lister *et al.,* 2009; Lister *et al.,* 2013). The DNMT3 family is involved in DNA methylation during germ cell development (Kaneda *et al.,* 2004; Reik, W., 2007). Then, DNA methyltransferase 1 (DNMT1) keeps DNA methylated throughout replication (Reik, W., 2007). It includes CG, CHG, and CHH. Plants' de novo DNA methylation occurs via the RNA-directed DNA methylation process. Many DNA methyltransferases retain cytosine methylation after DNA replication.

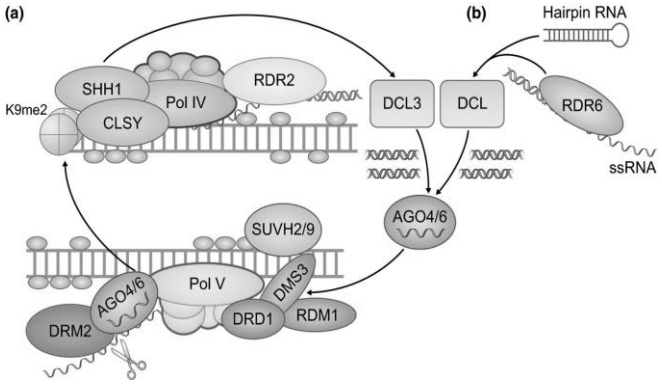

Figure 2. Methylation process in plants.

***DNA methylation for disease resistance in plants*:** Initially, DNA methylation is seen in plants throughout development. Plants show resistance to DNA viruses by allowing TGS of the virus's genome (Feng *et al.,* 2010; Rodriguez *et al.,* 2013; Wang *et al.,* 2014; Yang *et al.,* 2011). It now affects non-viral illnesses as well. The SA defense pathway is controlled in tomato DC3000 (PtoDC3000) (Yu *et al.,* 2013). The presence of active histone marks at the PR1 promoter in Pol V mutants suggests that chromatin is generated for rapid transcription against pathogens. PR1 expression was strong in the met1 nrpd2 mutant (Dowen *et al.,* 2012, Yu *et al.,* 2013). Cell death within and around the leaf's secondary veins was observed. Bacterial growth in Arabidopsis leaf vasculature is reduced in mean ddc and met1 nrpd2 mutants (Yu *et al.,* 2013). The loss of DNA methylation pathways is produced by eliminating CHH, CGCG/CHH, or CG/CHG methylation—the SA-dependent defense response eventually inhibits biotrophic pathogen proliferation. The antibiotic 5-azadeoxycytidine increases resistance to Xanthomonas oryzaepv (Akimoto *et al.,* 2007). Arabidopsis DNA is methylated in three ways: CG, CHG, and CHH (where H stands for A, T, or C) (Law, JA, and Jacobsen, 2010). However, non-CG methylation has been identified in specific cell types such as embryonic stem cells and brain cells (Lister, R. *et al.,* 2009; Varley, K.E. *et al.,*

2013). DNA methylation has been investigated in Arabidopsis. DNA methylation analysis of five-week-old Arabidopsis seedlings revealed a 24.7% methylation level in CG, CHG, and CHH sequence contexts, respectively (Cokus *et al.,* 2008). A high degree of methylation at most cytosine sites indicates that Arabidopsis CG methylation is maintained sufficiently after DNA replication (Lister *et al.,* 2008). The soil pathogen Agrobacterium tumefaciens suppresses crown gall tumor development through DNA methylation (Gohlke *et al.,* 2013). Surprisingly, integrated oncogene transcriptional gene silencing slows tumor development. Despite low methylation levels in wild-type tumors (Gohlke *et al.,* 2013), increasing methylation in plant genes promoted tumor development. Enzymatic DNA methylation reveals essential information about a gene's function via type, quantity, and location. DNA methylation is an epigenetic gene expression regulation mechanism that is inherited. It depends on the content of CG, CHG, and CHH sequences and surrounding genomic features. The methylation patterns obtained by bisulfite sequencing were compared to methyl filtering in McrBC+ Escherichia coli strains (Palmer *et al.,* 2003). It turns out that areas with high methylation are like those with the most repeats and fewest genes on each chromosome (Schnable *et al.,* 2009). Symmetric CG and CHG are more methylated than asymmetric CHH. Several studies have shown that specific centromeric repeats are unmethylated (Palmer *et al.,* 2003).

***Plantinnate Immune System*:** Plants lack an adaptive immune system. Plants also lack immunological memory and self-tolerance. The first line of defense for plants is a thick bark and a waxy cuticle. Hard shells, thorns, and spines protect plants against herbivores. Abrasions may weaken external defenses, and it leads to pathogen entry. Dead plants must move to the second line of defense. Like toxins and enzymes. These compounds poison pathogens and herbivores equally. Plant pathogens have several ways to survive.

Pathogenic bacteria enter through water/gas pores (stomata/hydathodes) or wounds and multiply in intercellular gaps (the apoplast). Aphids and nematodes feed by piercing plant cells. It can extend hyphae on top of, between, or through epidermal plant cells. Pathogenic and symbiotic fungi and oomycetes can invade host cell plasma membranes and feed structures (haustoria). Intimate interactions between the extracellular matrix, Haustoria, and host plasma membranes influence the result. Pathogens secrete virulence factors into plant cells to promote microbial fitness. Plants fight illness via barriers, secondary metabolites, and antimicrobials. Plants have various defenses against illness.

Along with secondary metabolites, plants produce antibacterial chemicals, proteins, and enzymes. Stomata can be closed to keep illness out, and plants use a hypersensitive response that causes cell death to fight infection. However, endophyte aid occurs when the roots produce chemicals that attract other beneficial bacteria.

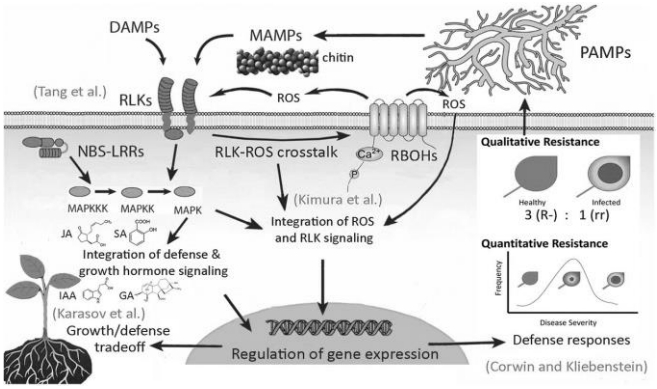

Figure 3. Simplified model of plant innate immunity.

***Plants Genomicinnate Immunity*:** Plant pathogens cause an immunological response by producing effector proteins. Plant genomes carry genes that determine which effectors induce immunity in particular species (ETI). Plants can either act directly by detecting pathogen virulence factors (effectors) or indirectly by monitoring host proteins that have been changed by effector activity. PAMPs are the first active defense (pathogen-associated molecular patterns). Pattern-recognition receptors (PRRs) on the cell surface receive PAMPs or

Table 1. Summary of techniques for manipulating DNA methylation in plants using programmable DNA-binding proteins.

Organism	Activity	DNA-binding protein	Effector	Reference
Plants	Methylation	ZF	SUVH9	Johnson *et al.,* (2014)
Plants	Methylation	ZF	SHH1	Gallego-Bartolome *et al.,* (2019)
Plants	Methylation	ZF	NRPD1	
Plants	Methylation	ZF	RDR2	
Plants	Methylation	ZF	MORC6	
Plants	Methylation	ZF	MORC1	
Plants	Methylation	ZF	DMS3	
Plants	Methylation	ZF	RDM1	
Plants	Methylation	ZF-SunTag	DRM-CD	Gallego-Bartolome *et al.,* (2019); Papikian *et al.,* (2019)
Plants	Demethylation	ZF-SunTag	TET1-CD	Gallego-Bartolome *et al.,* (2018)

MAMPs (microbe-associated molecular patterns) (Jones JD and Dangl JL., 2006). PAMPs trigger an immunological response, resulting in basal immunity (Boller T and Felix G., 2009). The pathogens then produce effectors that block PTI inside the host cell, causing microbial growth (Deslandes L and Rivas S., 2012; Dou D and Zhou JM., 2012). As a result, pathogen effectors can induce disease resistance. Resistance proteins will produce ETI (effector-triggered immunity) (Jones JD and Dangl JL., 2006; Maekawa *et al.,* 2011). Significantly, both PTI and ETI activation require massive transcriptional reprogramming, tightly regulated by chromatin-based regulatory mechanisms (Navarro *et al.,* 2004; Pandey SP and Somssich IE., 2009, Rushton *et al.,* 2010).

Conclusion: Considerable progress has been made in developing techniques for analyzing epigenomic data; however, several challenges remain. DNA methylation regulates gene expression. They do so by releasing effector proteins. Plant genomes carry genes that determine which effectors induce immunity in particular species (ETI). PAMPs are the first active defense (pathogen-associated molecular patterns). Pattern-recognition receptors (PRRs) on the cell surface receive PAMPs or MAMPs (microbe-associated molecular patterns) (Jones JD and Dangl JL, 2006). Pattern-recognition receptors identify conserved microbial fingerprints. This recognition activates an immune response. Methylation is an epigenetic alteration that happens in DNA bases. It is found at the fifth carbon of the cytosine pyrimidine ring in mammals and plants. Methylation may inhibit transposons, affecting gene transcription. Stressor-induced DNA methylation changes are dynamic. They may affect the TE expression of defense genes associated with TEs/repeats. Plants have cytosine methylation in all cytosine sequences, including CG, CHG, and CHH. Stress-induced DNA methylation modifications can affect the expression of TEs or defense-related genes linked with them.

Toxic stress-induced DNA methylation changes may be due to tissue-specific DNA methylation modifications, such as in leaf vascular DNA. It might be due to the leaf vasculature expressing RdDM factor genes and ROS1 before elicitation/infection. Pathogen stress is remembered through generations. However, whether this occurs in DNA methylation or other heritable chromatin changes is unclear. Agrobacterium tumefaciens, a soilborne biotrophic pathogen, causes crown gall tumors in Arabidopsis (Gohlke *et al.,* 2013). ETI is a quicker and more robust version of PTI (Thilmony *et al.,* 2006). Purifying selection is likely to be used on effectors that modify host targets biochemically (Rohmer *et al.,* 2004).

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